ISSN: 2634 - 1255

Journal of Gynecology Research Reviews & Reports

SCIENTIFIC Research and Community

Research Article

Open Access

Cerebral Palsy is 25 or More of Hypoxia Index and No Cerebral Palsy is 24 or Less of Index in Fetal Monitoring

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ABSTRACT

Fetal outcome was ominous if fetal heart rate (FHR) was late deceleration (LD) in the past, while 3 connected typical LDs were normal, and repeated LDs for 50 min were heavy fetal brain damage. Also, LD is defined as LD when it is repeated for 15 minutes. As the fetus is damaged by repeated hypoxic decelerations followed by cerebral palsy, but not by its late appearing in LD, novel fetal hypoxia index (HI) is the sum of all deceleration durations (min) divided by the lowest FHR (bpm) and multiplied by 100 in fetal monitoring. The hypoxia index was 25 or more in all of 6 cerebral palsy cases, while it was 24 or less in all 16 cases of no cerebral palsy. As error probability is almost zero in the chi2 test of hypoxia index, no cerebral palsy is decided when the hypoxia index is 24 or less, while it is cerebral palsy, if hypoxia index is 25 or more. The hypoxia index is adopted to all FHR decelerations and bradycardia, as hypoxia index does not evaluate the late appearing of deceleration, instead of past subjective deceleration pattern diagnosis in fetal monitoring.

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Received: July 16, 2020; **Accepted:** July 22, 2020; **Published:** July 29, 2020

Keywords: Fetal Heart Rate, Late Deceleration, Hypoxia Index, Cerebral Palsy, Fetal Monitoring

Introduction

FHR decelerations were classified into early, late, mild & severe variable ones in FHR patterns, where late deceleration (LD) was ominous but the author experienced controvercy facts, where 3 connected typical LDs resulted fully normal outcome with Apgar 9, while trequently repeated LDs for 50 min (Figure 1) resulted severe asphyxia and heavy brain damage. In addition, there was a definition, where LD should be diagnosed after 15 minutes repetition [1,2]. Thus, it was concluded that LD is ominous not due to its particular late appearing pattern, but ominous due to its frequently repeated decelerations, because fetal hypoxia develops FHR bradycardia, where the vagal nerve center of medulla oblongata is stimulated by hypoxia, that was confirmed by the linear relation of heart rate to arterial PaO_2 lower than 50mmHg and human fetal PaO_2 is 50 or less mmHg [2,3].

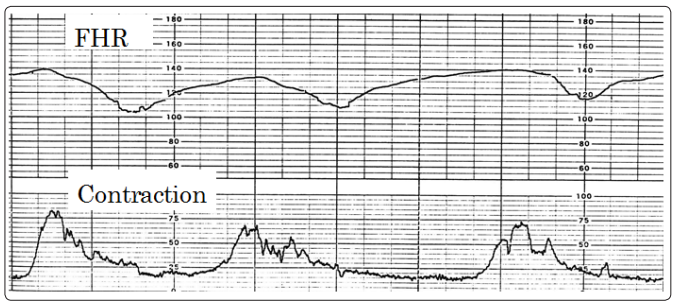

Figure 1: Caesarean delivery was refused, 50 minutes LD repetition and the loss of variability. Hypoxia index 26. Apgar score 3. Severe brain damage

The author studied the mechanism to develop LD in past reports, where the developing mechanism of LD was the compression of maternal pelvic iliac artery with contracted uterus prior to the FHR deceleration with lag time, while the LD disappeared by the change of maternal posture to lateral one removing the pressure of contracted uterus [2-4]. There was no reason to believe the presence of fetal own intrinsic pathology.

Methods

Transient bradycardias appear simultaneously at the uterine contractionsto push out the fetus in the labor regularly and repeatedly in fetal delivery,which is very regular uterine contractions similar to electric oscillation withthe same intensity and regularity, which may suggest thepresence of of apositive feed-back system composed of uterine excitation – uterus brain nerve – excitation of hypothalamus – excretion of contraction : oxytocn – circulation to uterus – enhancing the contraction. The function became strong and stable to develop strong contraction to push out the fetus. Thephenomenon of labor contraction developed by the regular excitation ofpositive feed-back system. The oxygen content in the circulating blood is insufficient and transient hypoxia develops, which stimulates parasym- pathetic center to develop transient bradycardia, which forms transientbradycardia and various decelerations, including periodic early and latedecelerations as well as mild and severe variable decelerations. The latedeceleration is particular mechanism, namely, contracted uterus pressesiliac artery to stop placental circulation and developing late hypoxia thenlate deceleration repeatedly, thus changing posture to lateral one takes off compression of uterus to recover placental circulation and forms nodeceleration (LD).The stopping placental circulation developed hypoxiarepeatedly developed and transient bradycardia, which causes pathologic hypoxic damages of fetal brain followed by cerebral palsy, while raredecelerations do not damage the brain

but terrified the author. Repeated delayed FHR deceleration after uterine contraction in LD was reported in past literatures, where the contracted uterus in the labor compressedmaternal pelvic iliac artery to stop placental circulation causing fetal hypoxia, which was shown in pelvic angiography in the labor, wherethe fact was that LD damaged fetal outcome by its frequent repetition, butnot by its particular late appearance, so that, novel hypoxia index is the sum of deceleration durations (min), divided by the lowest FHR (Figure 2), and multiplied by 100 to keep it integer [1-5].

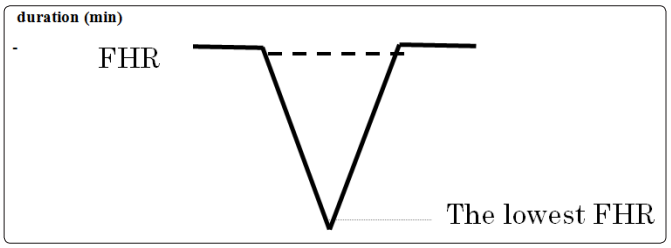

Figure 2: The determination of deceleration duration (min) at -10 bpm from the baseline and the lowest FHR (bpm) in hypoxia index

As the hypoxia index incorporates no lag time in the calculating equation,it is applied to all kinds of decelerations, namely, the application to all of early, late and mild or severe variable decelerations and even continuous bradycardia. Cases of 6 cerebral palsy cases and 16 no cerebral palsy, who were diagnosed in pediatric clinic, were collected and intrapartum hypoxia index was calculated by studying preserved intrapartum FHR records by the author under blind condition. This study was allowed by the ethical committee of Seirei Mikatahara hospital (No. 18-28).

Results

The technique to evaluate FHR changes was hypoxia index, and the target disease was infantile cerebral palsy whre no reduction was blamed in Dublin RCT by people to hope marvelous effect of fetal monitoring. This report is the first trial to predict fetal damage with numeric figure threshold. Present problem was to numerically reduce cerebral palsy, which has been achieved subjectively by experts of fetal monitoring with continuous observation of FHR record to reduce cerebral palsy with early caesarean delivery [6,7].

We collected 6 cases of cerebral palsy and 16 cases of no cerebral palsy, diagnosed in pediatric clinic. The FHR recorded in fetal monitoring were preserved in obstetric ward which were analyzed with novel hypoxia index by corresponding author.

The hypoxia index was 25 or more in all of 6 cases of cerebral palsy, and 24 or less in all of 16 no cerebral palsy cases (Table 1). The number of cerebral palsy was analyzed with χ^2 test, where p = 0.000008, significant difference, and the probability to make error was almost zero, so that we decided that cerebral palsy is prevented if the hypoxia index is 24 or less, and the cases whose hypoxia index is 25 or more can receive early cerebral palsy therapy.

Table 1: Comparison of cerebral palsy cases in high and low Hypoxia Index cases. There was significant difference of the cerebral palsy cases between groups of 25 or more and 24 or less hypoxia index

Hypoxia Index	Cerebral Palsy	
	Yes	No
25 or more	6 cases	0
24 orless	0	16 cases

$\chi 2$ test of cerebral palsy cases: p=0.000008, significant difference

Discussion

Compression of Blood Vessels:

There are effective procedure to change maternal supine posture to lateral ones as follows;

1. Supine hypotension
   Inferior vena cava was compressed by contracted pregnant uterus reducing the blood return to maternal heart developing maternal hypotension followed by the reduction of placental arterial blood flow, developing fetal hypoxia and FHR deceleration. It is well known that the supine hypotension disappears after changing aternal posture to lateral one from the supine.
2. Umbilical cord compression
   As the arterial blood supply to fetal placental villi capillary reduces due to the compression of umbilical cord, fetal oxygenation reduces and fetal bradycardia appears due to hypoxia. Maternal posture change will be effective to remove the cord compression and fetal bradycardia.
3. Late deceleration
   Pelvic large iliac artery including uterine artery were compressed by contracted uterus in every uterine contraction repeatedly stopping placental arterial blood supply developing fetal hypoxia and transient bradycardia of every late deceleration, which was confirmed by pelvic arterial angiography (Poseiro effect). The LD disappeared after maternal posture change to lateral one [3].

The Loss of FHR Accelerations in Early Fetal Hypoxia

Since fetal brain reacts fetal movement developing FHR acceleration (transient tachycardia). As fetal brain function is suppressed in the hypoxia, FHR acceleration is lost against fetal movements (non-reactive FHR). Although the loss of acceleration seems reversible, and the baseline variability is preserved in non-reactive FHR, it should be careful as the most heavy fetal brain damage, which is the loss of FHR baseline variability followed by cerebral palsy, develops in 2 weeks after the loss of acceleration, thus, it may be reasonable to perform early caesarean delivery, when the acceleration is lost.

The loss of FHR Variability

Fetal outcome was ominous even in very mild LD by Hon, however, mild LD was recognized when FHR variability is lost [1]. As the loss of variability was the mostly severe fetal brain damage similar to anencephaly [6], as fetal brain dose not respond to minor fetal movements. Thus, fetal asphyxia is treated by early C-delivery before the loss of variability, that means to deliver the fetus when the hypoxia index is 24 or less, not after the loss of variability that is irreversible and develops cerebral palsy.

Causes of Fetal Hypoxia

Fetal Disorders

Congenital or acquired fetal diseases can develop fetal hypoxia and FHR deceleration, because the heart rate is parallel to PaO2 when it is lower than 50mmHg, common level of fetal arterial blood.

Extrinsic Causes

Low maternal blood pressure, maternal hypoxia, abnormal placental oxygen transfer function, e.g. infarction in preeclampsia, intervillous space fibrin deposit, umbilical cord compression or uterine hypercontraction are able to cause fetal hypoxia.

Compression of maternal and fetal vessels

Maternal Supine Hypotension

Maternal inferior vena cava is compressed by contracted pregnant uterus in maternal supine posture to cause maternal hypotension

by less blood return to heart and fetal hypoxia causing FHR deceleration, which disappears after maternal lateral posture.

Compression of Iliac Arteries

Contracted pregnant uterus in maternal supine posture stops material blood flow to placenta causing fetal hypoxia. It was confirmed by pelvic angiography. Fetal deceleration occurs after the lag time, namely, late deceleration (LD) appears, where the LD disappears after maternal posture change to lateral one [3].

Umbilical vessel compression

It appears in the cord prolapse, lower presenting cord, nuchal cord, compression by fetal body and so on. Maternal lateral posture is effective in conservative therapy. Heavy case receives caesarean delivery.

Management of FHR Decelerations

As maternal lateral posture rejects the pelvic vessel compression caused by contracted pregnant uterus in maternal supine posture, i.e. vessel compression is removed by maternal lateral posture increasing blood supply to the placenta. As the effect of lateral posture is revealed by quick disappearance of deceleration, the mother has to take lateral posture after recording deceleration in fetal monitoring not to increase hypoxia index [4]. Other cause should be checked, if the deceleration is still present after the lateral posture.

Tocolysis will be done in the Uterine Hypercontraction

Orcinoprenaline was effective to the LD in uterine hyper contraction in the old report [4]. Tocolysis is effective by terbutaline administration.

Early Delivery

Severe LD case is cured by early delivery with C-section before the loss of variability to prevent fetal brain damage, in case of no intrauterine therapy. C-section is performed before the loss of FHR variability, which is the sign of heavy fetal brain damage [8]. Timely C-section will be indicated by severe FHR changes, the loss of FHR acceleration, decreased variability, or when the hypoxia index was high as 20, as hypoxia index of cases to lose variability was higher than 25 [8].

Actocardiogram

The neonate was vigorous when fetal acceleration duration ratio to fetal movement burst duration (A/B ratio) was higher than 1.0 in repeated LDs, namely, fetal movement study was effective to estimate favorable outcome [9].

Possibility of Fetal/Neonatal Damage

Although FHR is parallel to the PaO_2 under 50mmHg and fetal bradycardia is recorded, it is not the direct sign of fetal damage, namely, the bradycardia is a neurologic excitation of parasympathic center, where xperimental bradycardia does not develop under anesthesia. Fetal brain is damaged when FHR variability is lost by hypoxia, namely fetal bradycardia may show only fetal environment, while hypoxic fetal brain damage is expressed by the loss of fetal brain response to fetal movement, i.e. FHR variability is fetal brain response to minor fetal motion therefore, the loss of acceleration to fetal movement is early sign of fetal brain suppression, while the loss of variability is advanced severe fetal brain damage, confirmed by actocardiographic studies [8,9]. Although the loss of acceleration may be reversible, the loss of variability is irreversible and followed by cerebral palsy, thus, the fetus should be delivered when the hypoxia index is less than 24 to prevent cerebral palsy. Therefore, the LD caused by the compression of pelvic vessels which recovered after posture change may not be fetal damage, if the hypoxia index is less than 24. Please be careful to the hypoxia index level keeping less than 23 is vigorous without brain damage, while high hypoxia index can be hypoxic repeated deceleration may be fetal damage if the LD lasted for several 10 min. Please be careful to hypoxia index level keeping it around 20 by holding lateral posture. The fetal damage could be estimated by the *hypoxia index*, which must be around 20 to protect the fetal brain from hypoxic damage, even in emergency caesarean section, with lateral posture before surgery. That is only an example, and the author hopes rapid cure of deceleration in its earliest stage, e.g. the cure from deceleration with lateral posture through whole stage of labor.

Computerized FHR Analysis

Actocardiogram is connected to a fetal monitoring computer. Main purposes are three rather complicated calculation as follows;

Hypoxia Index

Appearance of deceleration, present hypoxia index, remained time to 23 hypoxia index are reported. Main functions are to prevent cerebral palsy and fetal demise, covering all FHR deceleraions and bradycardia keeping the index at around 20.

FHR Score

Five min' analysis and FHR evaluation to prevent fetal death and umbilical arterial blood acidemia. Keep it below 20.

Pathologic Sinusoidal FHR

Detection of severe fetal anemia by sinusoidal to prevent fetal death Separation of benign physiologic sinusoidal FHR with frequency spectrum analysis

Report to the doctor

10 results obtained are directly and rapidly reported physician in charge by direct mail but not by paper. It is also recorded in electric patients record.

Conclusion

There would be several matters in FHR deceleration. Maternal posture change to lateral one should be tried after detecting deceleration on the report and chart. Hypoxia index should be lower than 20 every time to prepare sudden accident. Although final decision will be cesarean section, possible intrauterine therapy would be tried. Actocardiogram and the computer reports are good help.

References

1. Hon EH (1968) An atlas of fetal heart rate patterns. Harty Press, New Haven.
2. Caldeyro-Barcia R, Poseiro JJ, Mendez-Bauer C, Gulin LO (1967) Effects of Abnormal Uterine Contractions on Fetal Heart Rate in Labor 9-27.
3. Poseiro JJ, Mendez-Bauer C, Caldeyro-Barcia R, Poseet SV (1969) Effect of uterine contractions on maternal blood flow through the placenta. Perinatal factors affecting human development,Paho Advisary Committee 161-171.
4. Caldeyro-Barcia R, Magana JM, Poseiro, JJ et al. New Approach to the treatment of acute Intrapartum fetal distress. Perinatall factorsaffecting human development. Patho Advisary Committee, 1969, 248-253.
5. Teshima N (1993) Non-reactive pattern diagnosed by ultrasonic Doppler fetal actocardiogram and outcome of the fetuses with non-reactive pattern. Acta Obstet Gynecol Jpn 45: 423-430.

6. Tsuzaki T, Sekijima K, Maeda K, et al. The survey on the perinatal variables and the incidence of cerebral palsy for 12 years before and after the application of the fetal monitoring system. Acta Obstet Ginecol jpn 1990; 42: 99-105.
7. Maeda K, Noguchi Y, Utsu M, Masaji Utsu, Takashi Nagasawat (2012) Central computerized automatic fetal heart rate diagnosis with a rapid and direct alarm system. The Open Medical Devices Journal 4: 28-33.
8. Maeda K (2014) Modalities of fetal evaluation to detect fetal compromise prior to the development of significant neurological damage 40: 2089-2094
9. Maeda K (2016) Fetal Actocardiogram Analysis of Fetal Motion and Heart Rate, Jaypee Brothers, New Delhi. http://www.jaypeebrothers.com/pgDetails.aspx?cat=s&book_id=9789352501861